

CLAIMS

What is claimed is:

1. An immunoregulator obtainable from urine that regulates Th1, Th2 or both Th1 and Th2 cell activity.
2. An immunoregulator obtainable from urine that modulates dendritic cell differentiation.
3. The immunoregulator of claim 1 that modulates dendritic cell differentiation.
4. The immunoregulator of claim 3 wherein said urine is obtained from a pregnant mammal.
5. An immunoregulator comprising an active component, or a functional fragment thereof, obtainable from a mammalian chorionic gonadotropin preparation, wherein said active component stimulates splenocytes obtained from a non-obese diabetes (NOD) mouse.
6. An immunoregulator comprising an active component obtainable from a mammalian chorionic gonadotropin preparation, wherein said active component protects a mouse against a lipopolysaccharide induced septic shock.
7. The immunoregulator of claim 5 wherein said active component is present in a fraction which elutes with an approximate molecular weight of 15 to 58 kilodaltons as determined in gel-permeation chromatography.
8. The immunoregulator of claim 5 wherein said active component is present in a fraction which elutes with an approximate molecular weight of 1 to 15 kilodaltons as determined in gel-permeation chromatography.
9. The immunoregulator of claim 5 wherein said active component is present in a fraction which elutes with an approximate molecular weight of less than one kilodalton as determined in gel-permeation chromatography.
10. The immunoregulator of claim 9 wherein said mammalian chorionic gonadotropin preparation is derived from urine.
11. The immunoregulator of claim 10 that regulates Th1, Th2 or both Th1 and Th2 cell activity.
12. The immunoregulator of claim 11 that modulates dendritic cell differentiation.

13. The immunoregulator of claim 12 wherein said stimulated splenocytes delay the onset of diabetes in a NOD-severe-combined immunodeficient mouse reconstituted with said splenocytes.

14. The immunoregulator of claim 13 wherein said active component inhibits gamma-interferon production of splenocytes obtained from a non-obese diabetes (NOD) mouse.

15. The immunoregulator of claim 14 wherein said active component stimulates interleukin-4 production of splenocytes obtained from a non-obese diabetes (NOD) mouse.

16. The immunoregulator of claim 15 wherein said active component reduces ASAT plasma levels after or during organ failure.

17. A method of treating an immune-related disorder in a subject believed to be in need thereof, said method comprising:

administering to the subject an amount of an immunoregulator obtainable from mammalian urine, wherein said immunoregulator modulates Th1, Th2 or both Th1 and Th2 cell activity and is administered in an amount sufficient to modulate the immune-related disorder.

18. The method according to claim 17 wherein said immune-mediated disorder is selected from the group consisting of chronic inflammation, diabetes, multiple sclerosis, and chronic transplant rejection.

19. The method according to claim 17 wherein said immune-mediated disorder is selected from the group consisting of acute inflammation, septic shock, anaphylactic shock, and acute or hyper acute transplant rejection.

20. The method according to claim 17 wherein said immune-mediated disorder is selected from the group consisting of auto-immune disease, systemic lupus erythematosus, and rheumatoid arthritis.

21. The method according to claim 17 wherein said immune-mediated disorder is selected from the group consisting of allergy, asthma and parasitic disease.

22. The method according to claim 17 wherein said immune-mediated disorder is selected from the group consisting of an overly strong immune response directed against an infectious agent, a virus and bacterium.

23. The method according to claim 17 wherein said treatment comprises regulating relative ratios, cytokine activity or both relative ratios and cytokine activity of lymphocyte subset-populations in a treated individual.

24. The method according to claim 23 wherein said subset populations comprise Th1 or Th2 cells.

25. The method according to claim 17 wherein said immunoregulator comprises an hCG preparation or a fraction derived thereof.

26. A pharmaceutical composition comprising an active component, or derivative thereof, obtainable from urine that stimulates splenocytes obtained from a non-obese diabetes (NOD) mouse, said stimulated splenocytes delaying the onset of diabetes in a NOD-severe-combined-immunodeficient mouse reconstituted with said splenocytes.

27. The pharmaceutical composition of claim 26 wherein said active component inhibits gamma-interferon production or stimulating interleukine-4 production of splenocytes obtained from non-obese diabetes (NOD) mouse.

29. The pharmaceutical composition of claim 26 obtained from a pregnant mammal.

30. The pharmaceutical composition of claim 29 comprising a clinical grade hCG preparation or a fraction derived thereof.

31. A method for treating an immune-mediated disorder in a subject comprising:
administering to the subject at least one immunoregulator, said immunoregulator obtainable from mammalian urine, and having Th1 and Th2 cell regulating activity, said immunoregulator being administered in an amount sufficient to modulate dendritic cell differentiation.

32. The method according to claim 31 wherein said immune-mediated disorder includes diabetes.

33. The method according to claim 32 wherein said immune-mediated disorder includes sepsis.

34. The method according to claim 33 further comprising regulating relative ratios, cytokine activity or both relative ratios and cytokine activity of lymphocyte subset-populations in said subject.

35. The method according to claim 34 wherein said subset-populations comprise Th1 or Th2 cells.

37. A method for selecting an immunoregulator comprising determining therapeutic effect of an immunoregulator by subjecting an animal prone to show signs of septic shock to a urine fraction or fraction derived thereof, and determining the development of septic shock in said animal.

38. The method according to claim 36 wherein said therapeutic effect is further measured by determining relative ratios, cytokine activity or relative ratios and cytokine activity of lymphocyte subset-populations in said animal.

39. The method according to claim 38 wherein said therapeutic effect is further measured by determining enzyme levels in said animal.

43. The immunoregulator of claim 4 wherein said pregnant mammal is a human.

44. The immunoregulator of claim 6 wherein said active component is present in a fraction which elutes with an approximate molecular weight of 15 to 58 kilodaltons as determined in gel-permeation chromatography.

45. The immunoregulator of claim 44 wherein said mammalian chorionic gonadotropin preparation is derived from urine.

46. The immunoregulator of claim 6 that regulates Th1, Th2 or both Th1 and Th2 cell activity.

47. The immunoregulator of claim 6 that modulates dendritic cell differentiation.

48. The immunoregulator of claim 6 wherein said stimulated splenocytes delay the onset of diabetes in a NOD-severe-combined immunodeficient mouse reconstituted with said splenocytes.

49. The immunoregulator of claim 6 wherein said active component inhibits gamma-interferon production of splenocytes obtained from a non-obese diabetes (NOD) mouse.

50. The immunoregulator of claim 6 wherein said active component stimulates interleukine-4 production of splenocytes obtained from a non-obese diabetes (NOD) mouse.

51. The immunoregulator of claim 6 wherein said active component reduces ASAT plasma levels after or during organ failure.

55. A method for selecting an immunoregulator, said method comprising:
determining a therapeutic effect of an immunoregulator by subjecting an animal prone to show signs of diabetes to a fraction obtainable from urine,

determining the development of diabetes in the animal, and

selecting an immunoregulator based upon therapeutic effect in the animal.

56. The method according to claim 55 further comprising measuring the therapeutic effect by determining relative ratios and /or cytokine activity of lymphocyte subset-populations in the animal.

57. The method according to claim 56 wherein the therapeutic effect is further measured by determining enzyme levels in the animal.

58. The method according to claim 55 wherein the immunoregulator is present in a fraction of pregnant female urine that elutes with an apparent molecular weight of 58 to 15 kilodaltons as determined by gel-permeation chromatography.

59. The method according to claim 55 wherein the immunoregulator is present in a fraction of pregnant female urine that elutes with an apparent molecular weight of 1 to 15 kilodaltons as determined by gel-permeation chromatography.

60. The method according to claim 55 wherein the immunoregulator is present in a fraction of pregnant female urine that elutes with an apparent molecular weight of < 1 kilodalton.

61. A method for selecting an immunoregulator, said method comprising:
determining therapeutic effect of an immunoregulator by subjecting an animal prone to show signs of septic shock to a fraction obtainable from urine, and
determining the development of septic shock in the animal.

62. The method according to claim 61 further comprising measuring the therapeutic effect by determining relative ratios and /or cytokine activity of lymphocyte subset-populations in the animal.

63. The method according to claim 62 wherein the therapeutic effect is further measured by determining enzyme levels in the animal.

64. The method according to claim 61 wherein said immunoregulator is present in a fraction of pregnant female urine that elutes with an apparent molecular weight of 58 to 15 kilodaltons as determined by gel-permeation chromatography.

65. The method according to claim 61 wherein said immunoregulator is present in a fraction of pregnant female urine that elutes with an apparent molecular weight of 15 to 1 kilodaltons as determined by gel-permeation chromatography.

66. The method according to claim 61 wherein said immunoregulator is present in a fraction of pregnant female urine that elutes with an apparent molecular weight of < 1 kilodalton.